BOLDT (H. J.)

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Professor of Gynæcology in the N. Y. Post-Graduate Medical School and Hospital; Gynæcologist to St. Mark's Hospital and the German Poliklinik; etc.

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Abscesses within the thickened inflamed and hyperplastic masses of the pelvic peritonæum, more especially the broad ligament, are by no means rare occurrences. According to our modern views of suppuration we seek its source in a purulent salpingitis. If the fimbriated extremity allows the escape of a droplet of pus we comprehend the subsequent localized peritonitis, the purpose of which is to shut off the focus of suppuration from the rest of the pelvic or abdominal cavity by reacting with what is known by the term of plastic or formative inflammation around the focus of suppuration. Since we know that the presence of the staphylococcus or rather its ptomaines is a requirement to produce suppuration we consider this process an infectious one, the micro-organism being carried from without first to the endometrium and from here into the tubal thence into the peritoneal cavities.

The process known by the term of suppurative oöphoritis, leading to a partial or total destruction of one or both ovaries is not quite so common. Obviously this process in most instances arises from the contact of the ovary with a focus of suppuration in its immediate vicinity—the broad ligament. In cases where the ovary is only partly destroyed, we find it after extirpation or at a *post-mortem* examination bordering on an abscess cavity and changed to such an extent that it often is not recognizable to the naked eye. Often there is no difference in the appearance or consistency

of the remnants of the ovary and the adjacent considerably thickened pseudo-membranous masses of the broad ligament. Under these conditions we must resort to the microscope in order to be able to ascertain how much of the original ovarian structure is left; the main guides then being the tortuous arteries of the medullary portion of the ovary as well as the remnants of menstrual follicles.

I have come into possession by operation of some specimens, showing a marked degree of suppuration of the ovarian tissues which I propose to describe the more so since the manner in which inflammation and suppuration can be established has not yet been subjected to microscopical proof in the ovarian tissues.

Clinically I distinguish the abscesses which are of puerperal origin from those in which by a careful consideration of the history no such ætiological factor can be found so that necessarily we must speak of them as non-puerperal ovarian abscesses. The latter variety are comparatively rare, as can be seen by analyzing the cases published with care, when we will find facts in the history which will show that the origin is due to some pathological factor arising from the puerperium. By the term puerperium I include abortions and miscarriages as well as delivery at full term.

It is not my purpose to consider those conditions closely allied to our subject and frequently termed and described as ovarian abscess; I refer to suppurating ovarian cystoma and tubo-ovarian abscesses, these conditions are of frequent occurrence compared to true ovarian abscess. In the majority of cases the microscope becomes a necessary adjunct to determine the diagnosis.

The interstitial ovaritis giving rise to the abscess may be more or less remote from the respective puerperium hence its non-recognition from such puerperium. Gonorrhœa, operations on the cervix, and curettings are among the causes leading to the production of chronic or interstitial ovarian abscess.

From the above it follows that we have endometritis, non-suppurative salpingitis, parametritis or peri-oöphoritis accompanying the abscess; or that one or more of these conditions have at some time or another previously existed.

The symptoms of chronic suppurative ovaritis are chiefly those of pain, most intense in the ovarian region; from here it radiates in all directions, the lumbar and sacral region, hypogastric region, and occasionally along the course of the large nerves, the sciatic and crural, these pains depending greatly on the accompanying ovaritis, peri-ovaritis and parametritis. We may also have reflex nervous symptoms the more prominent of which are gastric disturbances

such as nausea and retching, but seldom vomiting. Cephalalgia is also frequently present. Physical examination reveals an ill-defined mass to one side or the other of the uterus which is according to the extent of the parametritis present, more or less movable—it is exceedingly painful to touch and varies in size according to the size of the abscess cavity—the latter in such cases varies from the size of a hazelnut or smaller to that of an English walnut, containing from two grammes to six or eight grammes of pus. The patients are emaciated, have a poor appetite and are anæmic. The temperature is sometimes normal, at others, in the same patient elevated from 0.5° to 2° F.

The pain differs from that of marked salpingitis or salpingo-ovaritis by the fact that it is independent of the menstrual period. It is a steady pain, sometimes dull, sometimes lancinating or piercing; if intermissions occur they are brief, lasting not longer than a few hours. It is necessary to distinguish the enlarged ovary separate from the tube if the diagnosis is to be made with any degree of certainty. The examination is more satisfactory under anæsthesia.

A positive diagnosis can, however, not be made at this period, but later when for some reason or another an acute process is implanted upon that which I have termed chronic, the diagnosis becomes easier—the small tumor increases in size, becoming as large as a hen's egg or larger; fluctuation is more or less perceptible and the pain is now more localized to the seat of the abscess; the area of radiation is now, judging from the voluntary statements of the patients limited to the lower part of the abdomen. The pain is without intermission and the febrile symptoms are marked. Temperature, slight chills, pulse and thirst are conspicuous in subacute suppurative oöphoritis.

The differential diagnosis lies between tubo-ovarian abscess and suppurating ovarian cystomata, and parametric abscess. From the former it can only be diagnosed if the tube can be separately felt and if we have had an opportunity to observe the patient for some time previously and then finding the change in the physical signs and symptoms already alluded to when an acute process is established upon the chronic ovarian abscess—the latter sometimes extending over a long period of time, viz., a year or more.

The same differential landmarks hold good for *small* suppurating ovarian cystoma; it is, however, an utter impossibility to be sure of the diagnosis unless the *development* has been watched. In parametric abscess the vaginal walls are more or less infiltrated and the mass felt feels firmer on the floor of the pelvis, and generally

there is a little bulging into the vagina. Ovaries, the seat of an abscess, are often very high up and adherent to the intestines which prevents their prolapse. The prognosis as to life in chronic ovarian abscess is not bad, the patient, however, remains an invalid as long as the condition remains, but the moment an acute condition supervenes a different aspect is present. It is very fortunate for the patient in case of spontaneous rupture under such circumstances that this takes place into the intestine, which is most frequently the case if such occurs, owing to adhesions to these, but the great danger is that rupture takes place intra-peritoneally as was the case in one of my patients, and owing to the extreme virulence of the pus, the patient is bound to die from a rapid septic peritonitis perhaps before she has fairly recovered from the shock of rupture, as we see it sometimes in an intra-peritoneal rupture of a peri-typhlitic abscess. That there is always danger of such rupture everyone will recognize.

Treatment. When the ovarian abscess is small so that the diagnosis is doubtful on account of the lack of sufficient physical signs, those of an ordinary ovaritis or peri-oöphoritis predominating, it is justifiable for one to use local treatment for a short time, but when it is seen that the patient does not improve except perhaps temporarily, then the proper procedure to carry out, both to relieve the patient and to avoid subsequent danger, is to open the abdomen and to remove the offending organ.

The case is different with a woman suffering with sub-acute ovarian abscess. Here the diagnosis can be made with greater probability—*with positiveness that a pathological condition is present which demands an operation*—without such operation the patient is in the dangerous position spoken of previously. The methods of operating should differ. If the walls of the abscess are thin and the gland is adherent, the contents should be aspirated before removal is attempted, no matter whether the tumor be only as large as a hen's egg or whether it be larger, so as to avoid the danger of rupture during enucleation and thus the contamination of the peritoneal cavity with the pus, the character of which one cannot be positive of beforehand, but it is usually extremely virulent. Dr. R. Schaefer, of Berlin, (see Zeitschrift f. Geburtshülfe und Gynäk., vol. xx, part 2, page 281–282) has made some interesting bacteriological researches with the pus obtained in such cases and in pyosalpinx, which at once demonstrate the reason why in many cases of the latter, if the pus enters the abdominal cavity, the patients recover, and why in others they die. The streptococcus pyogenes

brings the danger. In another class of cases in which the ovarian abscess has reached a much larger size, the patient's general condition poor, and the sac so firmly adherent to intestines and pelvis that complete removal seems either impossible or very hazardous, if in such case one can satisfy himself that the sac can be reached per vaginam, which is not difficult after the abdomen has been opened, no attempt should be made for obvious reasons, but the opening should be made from the vagina, the trocar being guided with the aid derived from the abdominal incision, a drainage tube inserted and the case treated like an ordinary pelvic abscess. The diagnosis that the respective case was really a true ovarian abscess can of course not be confirmed by anatomical proof, unless the patient should die, which I think is not probable if care has been exercised in every step of the operation. I deem it very doubtful that such cases as have been described as ovarian abscess as large as a man's head or even larger, really belong to that class and it stands to reason that they must be looked upon with suspicion when we consider the anatomy of the ovary.

My experience is based upon the following undoubted cases of non-puerperal ovarian abscess:

Case I.—Published in *American Journal of Obstetrics*, vol. 21, p. 511. *Intra-peritoneal Rupture of Ovarian Abscess and Death.* The unusual features of this case were that none of the symptoms were present which we should expect to find; the case would indeed permit doubts had it not been anatomically proven to belong in the category under consideration.

Case II.—No. 665, vol. 2., æt. thirty-eight; married fifteen years; four children. No abortions nor miscarriages. The last child was born nine years ago. All the labors were normal; menstruation also normal. There were no symptoms causing the patient to complain until in the spring of 1886, when she commenced to complain of a dull pain in the left ovarian region, which gradually grew worse and radiated to the hypogastrium and sacral region. When seen in 1887, perimetritis, oöphoritis and peri-oöphoritis was diagnosed. The patient was lost sight of until early in 1889; the pain at that time had become much more severe and almost constant, frequently lancinating, and increased in severity on riding or walking. Associated there was peculiar drawing pain on the outer side of the thigh. Micturition was painful, and sometimes tenesmus. Examination showed the uterus in physiological anteflexion, somewhat sensitive on bimanual examination. To the left of the uterus a mass exceedingly tender to touch, somewhat mobile, about the

size of a small hen's egg could be felt; its outlines were irregular. The diagnosis made formerly was reiterated plus the now more intense inflammation and probably salpingitis. Laparotomy in May, 1889, proved the correctness of the diagnosis and in the centre of the ovary an abscess was found containing about ten grammes of pus. Recovery uneventful.

Case III.—A——e O——z, æt. twenty-seven; married ten years; three children; one abortion at three-and-one-half months. The last child was born five years ago, the labor being completed with forceps. Recovery after each puerperium being undisturbed. She claims to have been perfectly well up to within five months of her coming under my care, when she commenced with pain in the left ovarian region, which radiated over the entire lower part of the abdomen; backache was present occasionally. Menstruation irregular in intermissions of two-and-one-half to three weeks, the flow lasting two days; no increase of pain. Occasionally the patient had slight chills and headache; nausea and retching at long intervals. Examination revealed the uterus anteposed; to the left a slightly fluctuating mass, firmly adherent, encroaching upon the uterus but separate from it. The tumor could be moved sufficiently to show its independence of the uterus. On the right side the remnants of a former parametritis were felt; posterior there was chronic perimetritis. Diagnosis, tubo-ovarian abscess or a small suppurating ovarian cystoma. Operation and subsequent examination in June, 1889, showed the tumor felt to be an ovarian abscess holding about three grammes of pus. Recovery was undisturbed.

Case IV.—J. B., æt. twenty-six; married two-and-one-half years; never pregnant. menses have been regular until two weeks prior to admission into the hospital, since then she has been constantly flowing. History of more or less constant pains in the right ovarian region, at the establishment of the menstrual flow, however, the pain would cease. Constipation and painful micturition.

On examination the uterus is found anterior and slightly to the left of the median line; to the right of the uterus a fluctuating tumor is felt which is diagnosed to be tightly adherent to the floor of the pelvis, the mobility of the tumor is consequently almost *nil*; it feels to be as large as a uterus at the second month of gestation. The temperature fluctuates between 99° and 101° F. the higher temperature being present, evenings. The diagnosis rested between a small suppurating ovarian cystoma and an ovarian abscess.

At the time of operation (November 8, 1891), it was found that the adhesions to the intestines, omentum and pelvic cavity were un-

usually dense. The tumor was aspirated, still all the pus was not emptied by this procedure, as on enucleation the sac tore a number of times and the peritoneal cavity was contaminated with the foul pus contained in the tumor, the sac—an ovarian abscess—contained about 150 to 180 grammes of pus. This is the largest ovarian abscess which I have seen. The patient died of septic peritonitis on the second day subsequent to operation.

A fifth case about which there is no anatomical report, I refrain from reporting.

The following cases operated upon by me I give as examples of cases which I would consider to come under the head of puerperal ovaritis.

E. S—n, æt. thirty, married twelve years; three children and two abortions; the last abortion ten years ago without any known cause; the last child died fifteen months ago. Ill seven months. Although her confinements and abortions were from description normal, by careful interrogation it was elicited that leucorrhœa existed from the birth of her last child, and that at times there was some pain in the left inguinal region, but it was inconstant, only being present on extreme physical exertion and sometimes after intercourse. Only during a period of seven months she was in constant pain in the left ovarian region. The diagnosis of perimetritis, ovaritis and salpingitis having been made and not the slightest improvement from treatment resulting, I opened the abdomen and removed the respective adnexa. A complete cure resulted; it was shown that the persistency of the symptoms were due to a small abscess in the centre of the ovary.

C. K——t, æt. thirty, married thirteen years, had two children; the last ten years prior to her consultation with me; no abortions nor miscarriages; labors normal. Had been suffering nearly constantly the last eight years with pain in the left ovarian region and backache. Occasionally, however, she also had pain in that region during the intervening two years after her last confinement, and always one week prior to menstruation. On operation, besides the salpingitis, a small abscess was found in the enlarged ovary.

C. F——n, æt. twenty-six; married seven years; two children, the last one year previous to her consultation. One abortion caused by physical exertion at four-and-one-half months' gestation, four-and-one-half months previously. Her confinements were normal. Since the abortion she has constant pains in the right umbilical region; on standing erect, pain in the hypogastrium; flatulence, intense dyspeptic symptoms, pain in both ovarian regions. Bowel

and bladder disturbances, menstruation irregular. Perinæum and cervix lacerated; the uterus is a little to the left of the median line; on the right side of it a mass is felt the size of a goose-egg, sensitive to pressure. Diagnosis: salpingo-oöphoritis, perimetritis dextra. On operation it was found that the tube was in a state of catarrhal inflammation. The much-enlarged ovary contained an abscess, the cavity of which was as large as a walnut. Histologically no difference exists between this variety of ovarian abscess and the chronic non-puerperal suppurative ovarian inflammation.

Immediately after removal the thickened masses were placed into a one-half-of-one-per-cent. solution of chromic acid, the pus cavities having been previously emptied. Those pieces which were suspected to be remnants of the ovaries, were cut out imbedded in celloidine and sliced into sections in the usual manner. According to the different tissues involved I propose to describe them under the following headings:

Fibrous Connective Tissues.

The inflammatory process of this tissue is best studied in the medullary portion of the ovary, so conspicuous by the presence of tortuous arteries. Unquestionably a large amount of bundles of smooth muscle fibres is mixed with the fibrous connective tissue in the medullary portion also; but the course taken by the inflammatory process both in connective and muscle tissue is perfectly identical to such an extent that in the inflamed ovary we are at a loss to tell which is muscle and which connective tissue, except in the middle coats of the arteries, of which I will speak later on.

The tissue surrounding and accompanying the arteries, is found in the condition known as acute inflammation, which is more intense the nearer the border of the pus cavity. If we analyze a portion not very markedly inflamed we can readily satisfy ourselves in regard to the following points. The original delicate and interlacing bundles of fibrous connective tissue are first transformed into granular protoplasmic masses, evidently often a liquefaction of the basis substance proper. In places furthest away from the suppurative destruction, many bundles are seen unchanged, to wit: still possessed of their basis substance; while this substance disappears to a large extent in adjacent bundles. Between the bundles the original protoplasmic bodies, the so-called connective-tissue corpuscles are enlarged, coarsely granular and broken up into a varying number, into lumps, known by the term of inflammatory corpuscles. Not only do the protoplasmic bodies between the

bundles give rise to a varying number of such corpuscles, but also the protoplasm sprung from previous bundles. This protoplasm at first appears coarsely granular and splits up into a number of spindle-shaped, oblong, or globular bodies, the origin of which from previous bundles is shown by the fact that quite often only a certain portion of a bundle is split up into inflammatory corpuscles while another portion remains unchanged or at least recognizable as

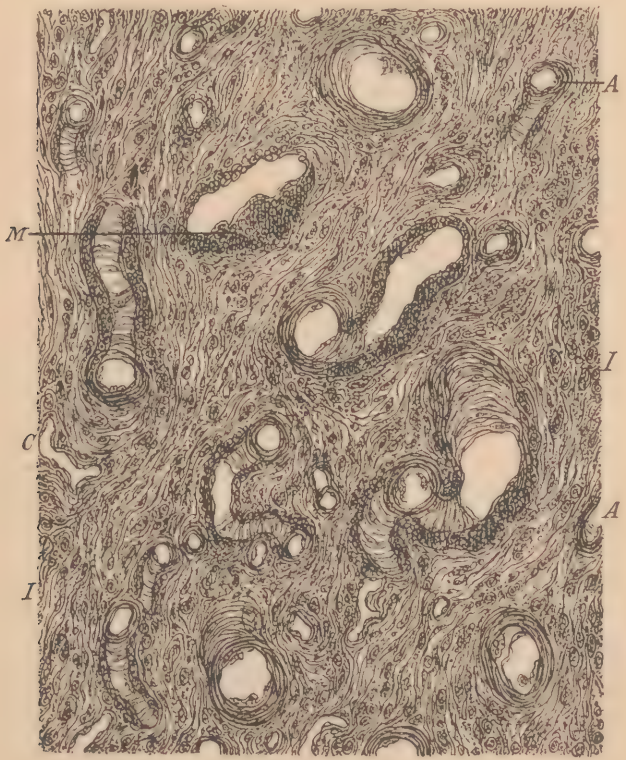

FIG. 1.

Intense acute oöphoritis. Medullary portion x 200. *AA* unchanged arteries. *M* middle coat of artery transformed into inflammatory corpuscles. *C* Capillary. *II* inflammatory infiltration of connective and smooth muscle tissue.

a bundle of fibrous connective tissue. The more the acute inflammation advances the less fibrous connective tissue remains visible in its original shape. Instead of it a new tissue is formed somewhat resembling adenoid or lymph tissue, or in the highest degree of

inflammation the original tissue is transformed into a mass of glistening globular corpuscles between which only scanty vestiges of fibrous connective tissue can be discerned. With higher powers of the microscope even such highly inflamed portions of the connective tissue prove to be a continuity, since all the inflammatory corpuscles are interconnected by means of delicate offshoots traversing the interstices between the inflammatory corpuscles. This interconnection of the corpuscles remains unbroken up to the border of the pus cavity, where the tissue forms an irregularly jagged, as if corroded, boundary line, beyond which there is no more tissue but only a mass of isolated granular and nucleated corpuscles known by the term of pus corpuscles. From my description it may be seen that I differ from the views of many pathologists that inflammation and suppuration are due to nothing but an emigration of colorless blood corpuscles or leucocytes from capillaries and small veins. I could satisfy myself that the main mass of inflammatory tissue is furnished by a previous fibrous connective tissue, after a liquefaction of its basis substance. As long as the newly-formed inflammatory corpuscles remain interconnected the original character of the tissue is unquestionably greatly altered; however, it still remains a tissue. It is only after a breaking asunder of the inflammatory corpuscles that the tissue is entirely destroyed, the corpuscles previously termed inflammatory being now pus corpuscles. Pus, therefore, is a broken-up tissue, which will never be tissue again.

Myxomatous Tissue.

The ovary affords an excellent opportunity to study the inflammatory changes of the myxomatous tissue in those portions which we know by the term of menstrual bodies. It is generally agreed, that the result of reparative tissue change after the rupture of a Graafian follicle is that the so-called structureless membrane of the follicle remains nearly unchanged and assumes a peculiarly convoluted or folded shape, remaining conspicuous by its high refracting power, its nearly homogeneous appearance and the capacity of taking up a deep-red stain after treatment with ammoniac and carmine. Residues of this part of the follicular wall are frequently met with in my specimens of acute and suppurative oöphoritis. The so-called structureless convoluted membrane remains nearly unchanged even though surrounded by the intensely inflamed cortical tissue of the ovary. This fact would prove that the so-called structureless membrane is allied to the elastic substance, being a

variety of basis substance of fibrous connective tissue and possessed of a high degree of resistance and indestructibility.

Menstrual bodies of a more recent date are characterized by the presence of a convoluted structureless membrane surrounding and

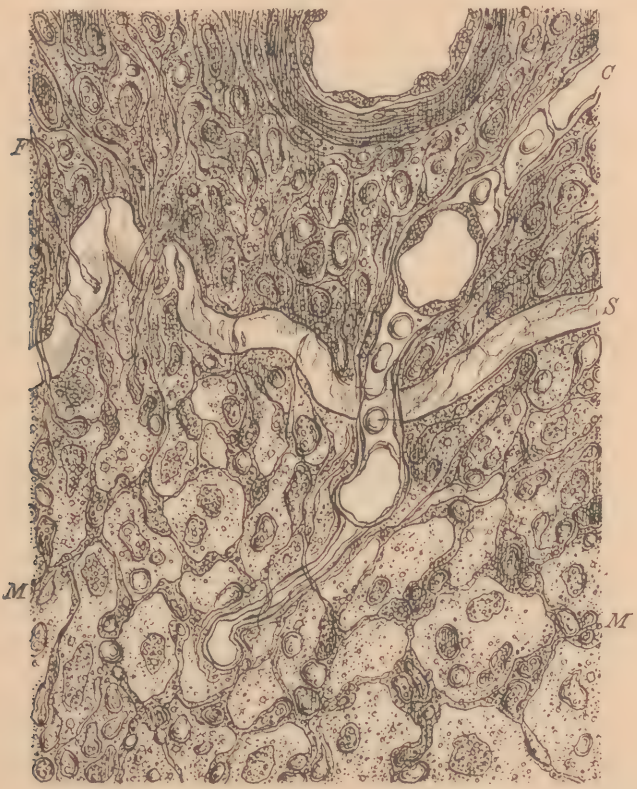

Fig. 2.

Intense acute oöphoritis, involving a myxomatous menstrual body x 500. *F* inflamed fibrous connective tissue. *MM* inflamed myxomatous connective tissue of a menstrual body. *S* so-called structureless membrane at the boundary of the menstrual body. *A* Artery in transverse section. *C* forming capillary blood vessel, with an offshoot into the myxomatous tissue.

enclosing a varying amount of myxomatous or mucoid connective tissue. This latter tissue often holds brown pigmented clusters indicative of hæmorrhage after bursting of the Graafian follicle and is, as a rule, destitute of blood vessels.

If now an inflammatory process invades the cortex of the ovary, the myxomatous tissue of the menstrual follicle likewise shares in the inflammation. We then observe a considerably augmented number ot protoplasmic bodies along the original myxomatous network. At the same time a variable number of protoplasmic bodies make their appearance in the meshes of the myxomatous tissue, which in a normal condition appears only finely granular or almost homogeneous. Nevertheless this basis substance, being a modified protoplasm must hold a certain amount of living matter which upon inflammatory irritation produces a varying number of protoplasmic bodies. The origin of the latter can be traced step by step from a coarse granule to a shining homogeneous globular lump and lastly to a granular nucleated body. Certain portions of the myxomatous tissue may appear little changed; others, however, will appear crowded with inflammatory corpuscles, the origin of which from the preëxisting myxomatous network as well as the myxomatous basis substance held in the meshes of the network, is an indisputable fact, since the tissue holds no blood vessels from which an emigration of leucocytes could have taken place.

The so-called structureless membrane often appears perforated in a radiating direction by oblong protoplasmic bodies, by means of which a continuity is established between the inflamed cortical tissue of the ovary outside and the myxomatous tissue inside the follicular wall. In many places this wall itself breaks up into numerous spindle-shaped bodies mostly in a radiating direction, whereby both high refraction and the deep-carmine stain are lost. This fact furnishes a strong proof of the viability of the follicular wall. Even this tough elastic substance will yield to an intense inflammatory process by becoming liquified and capable of proliferation.

Inflamed menstrual bodies not infrequently contain blood vessels, moving in the vicinity of the follicular wall. The blood vessels are capillaries and appear either empty or filled with variable number of blood corpuscles. In fig. 2 *C*, the manner in which newly-formed capillaries penetrate the myxomatous tissue is illustrated. It is done by an outgrowth of originally solid, later vacuolized and at last hollowed-out tracts of living matter, usually the lateral offshoots of already-existing capillary blood vessels.

In the figure the upper branch of the capillary still shows transverse bars in connection with the walls of the future capillary, which means vacuolation of an originally solid cord. One of the branches of the capillary vessel has pierced the follicular wall and

terminates in a club-shape within the myxomatous tissue, already exhibiting an extremely narrow calibre.

The final result of the inflammation of the myxomatous tissue is identical with that of fibrous connective tissue. It is transformed into a heap of inflammatory corpuscles with a complete disappearance of the follicular wall and a final breaking-up into pus corpuscles.

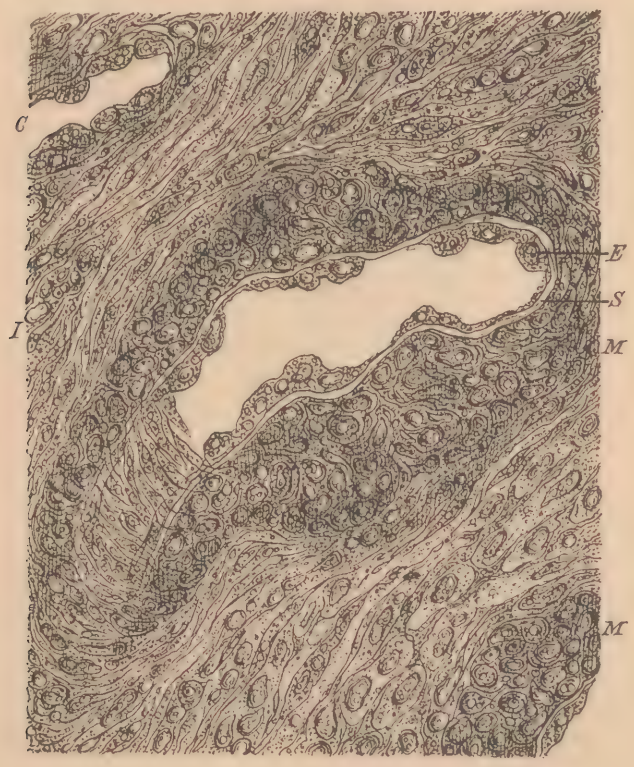

Fig. 3.

Arteritis in the medulla of an intensely inflamed ovary x 500. *E* endothelia coarsely granular. *S* so-called structureless membrane. *MM* smooth muscles of middle coat, breaking up into inflammatory corpuscles. *C* capillary blood vessel. *I* inflammatory infiltration of fibrous connective tissue.

Smooth Muscle Tissue.

As stated previously it is impossible to study the inflammatory changes of the smooth muscle tissue in the cortex of the ovary, since it is so closely mixed with fibrous connective tissue and the

result of the inflammation is so similar in both varieties that the source of the inflammatory corpuscles cannot be readily traced. The middle coat of the arteries, however, afford an excellent opportunity to study the myositis, since there cannot be the slightest doubt as to the situation of the smooth muscle tissue. See fig. 3.

Here we observe first an enlargement and coarse granulation of the nuclei of the smooth muscle fibres, most conspicuous in these longitudinal sections. Next the bodies of the spindles become coarsely granular and produce a number of glistening lumps, whereby the bulk of each spindle is noticeably augmented, the result is a broadening of the middle coat of the artery which at first is not uniform throughout the arterial wall. A number of inflammatory corpuscles will thus be produced either homogeneous of high refraction and small size, or nucleated and coarsely granular bodies will appear intermixed with glossy spindles, the origin of which is probably the interstitial connective tissue, the so-called paramysium. At the same time marked inflammatory changes take place in the endothelial lining of the artery. The endothelia become irregularly enlarged coarsely granular and their nuclei homogeneous and augmented both in bulk and number. This so-called elastic or structureless layer between the endothelia and smooth muscles seems to resist the destructive process longest. The final result of this form of arteritis is a breaking up of all constituent tissues into tracts and clusters of inflammatory corpuscles with a complete loss of the calibre and some vestiges of elastic membrane. Such formations are conspicuous by the ease with which carmine is taken up by them and the circumstance that they sometimes remain traceable in connection with little-changed arteries. In some of my cases there was a different kind of arteritis, *i.e.*, endarteritis obliterans. In this process it is mainly the endothelium which furnishes the inflammatory corpuscles leading to a more or less complete choking of the calibre. The final result is the appearance of fibrous connective tissue in the previous calibre, often with comparatively slight changes in the middle coat. I am unwilling to attribute this latter form of arteritis to acute oöphoritis leading to suppuration, since we know that it is a morbid process often met with in the ovaries and apparently independent of serious morbid changes of the latter. In the process of suppuration the tissue filling the centres of the arteries will break up into inflammatory corpuscles the same as the muscle coat and all vessels perish by being transformed first into inflammatory corpuscles and afterward disintegrate into pus corpuscles.

Epithelial Tissue.

Two pronounced epithelial formations are concerned in acute oöphoritis, viz., the Graafian follicles and the surface epithelium of the ovary. In one of my specimens I had an excellent opportunity to study the inflammatory changes of a Graafian follicle, somewhat advanced in development. See fig. 4.

The follicular wall appeared to be broken up into spindle-shaped bodies and inflammatory corpuscles, which undoubtedly had their origin from fibrous connective tissue. The shining epithelia of the follicle have become enlarged and irregularly shaped. Their nuclei had assumed a homogeneous glossy appearance and their number appeared augmented up to four or six, evidently in

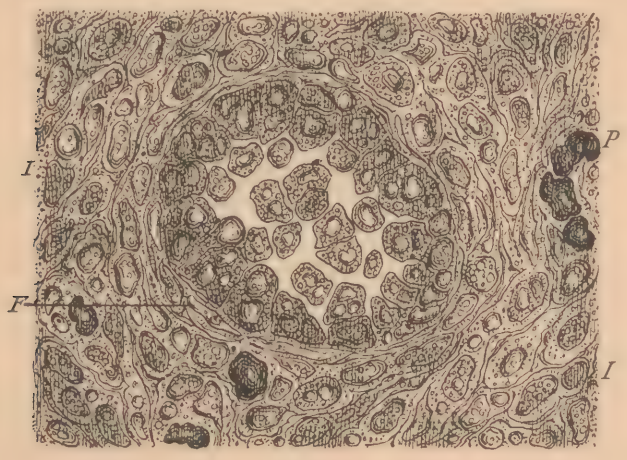

Fig. 4.

Suppuration of Graafian follicle x 500. *II* intensely inflamed corticle tissue around Graafian follicle. *P* pigment clusters from previous menstrual hæmorrhage. *F* wall of follicle.

consequence of their division. The protoplasm of the epithelia is coarsely granular and in some places there are distinct marks of division splitting up the protoplasm into pieces of varying sizes by means of delicate thorny projections. The smallest pieces generally have the average size of inflammatory corpuscles and it appears plain enough that the lining epithelium of a Graafian follicle breaks up into such corpuscles as does also the surrounding fibrous connective tissue. The section in the specimen presented went through the periphery of the follicle and I was unable to trace the

inflammatory changes of the yolk or the vesicular germinative. The surface epithelium of the ovary was found lining the abscess cavity in one of my cases. Here I could trace the row of the columnar epithelia from comparatively insignificant inflammatory changes up to their destruction into pus corpuscles in the situation mentioned. See fig. 5.

The most conspicuous change of the epithelia is their transformation into so-called mother-cells. We notice a number of vesicles holding a number of inflammatory corpuscles, aside from granules, *i.e.*, disintegrated protoplasm, which have probably sprung from the nuclei of the epithelia ; at the same time homogeneous

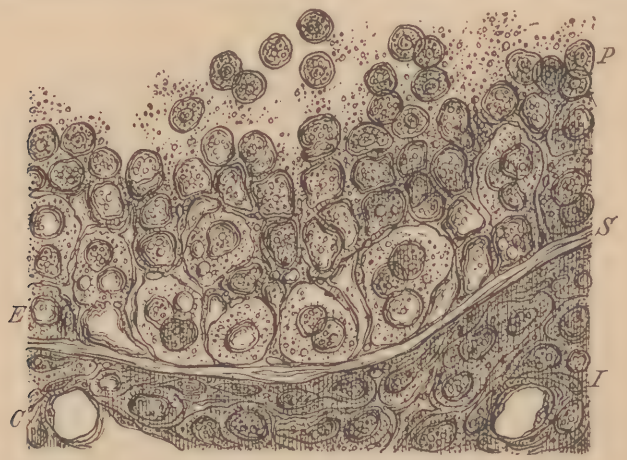

Fig. 5.

Suppuration of covering epithelium of ovary x 500. *S* so-called structureless membrane. *E* epithelia in intense inflammation. *P* pus corpuscles sprung from epithelia. *I* inflamed connective tissue. *C* capillary blood vessel.

spindle-shaped masses were seen bordering on the vesicles. The origin of such solid spindles seems to be the living matter traversing the cement substance between the epithelia. It has been shown by L. Elsberg that the so-called thorns uniting the epithelia may coalesce and produce solid spindle-shaped bodies. It seems mainly their presence which seems to cause the appearance of so-called mother-cells, which as we know to-day is due to an endogenous new formation of living matter within the epithelia. Where such a formation could not be seen, the epithelia to be broken into rows of inflammatory corpuscles, the arrangement of which together

with the absence of carmine stain so characteristic for epithelia generally, was indicative of their origin from epithelia. Rows of this description could be seen also in connection with so-called mother-cells, when they occupied the outermost portions of the previous epithelia. The constituent elements of the rows remain at first interconnecting but afterward the connections are broken, the inflammatory corpuscles detached and floating in an albuminous liquid they now bear the name of pus corpuscles.

I have given a description of the inflammatory changes of all constituents of the ovaries. The inflammation in the cases examined by me was intense and led to the production of abscesses. To my knowledge such changes have not been described. Still they are of great value to prove that inflammation and suppuration are not based on an immigration of colorless blood corpuscles, as had been asserted some twenty years ago by Cohnheim. My conviction is that all constituent tissues of the ovaries participate in an active manner in the production of inflammatory corpuscles which, being broken asunder furnish that which we know by the term of corpuscles.